TECHNICAL MEMORANDUM

Hanover/Whitewater Creek Investigation Unit – Human Health Risk Assessment

Chino Mines Company (Chino) is submitting this Technical Memorandum (TM) for the Hanover/Whitewater Creek Investigation Unit (HWCIU) in an effort to expedite activities in this investigation unit. This technical memorandum provides a human health risk assessment that includes hazard quotients (HQ) and cancer risk estimates for the HWCIU based upon data, equations and exposure parameters presented in the *Technical Memorandum: Risk-Based Analysis of Phase I Data for the Hanover/Whitewater Investigation Unit* (Neptune, 2001). Chino looks forward to the opportunity to discuss this memorandum with you further. Our rationale and methodology are provided briefly below.

The *Technical Memorandum* (Neptune, 2001) presented preliminary hazard indices (HIs)[1] and cancer risks for on-site residential (overbanks) and off-site residential (bars and active channel) for Exposure Reach 1 (E1); however, these estimates were not reported by individual metal or pathway. Section 4 of the *Technical Memorandum* provided a sensitivity analysis, but certain pathways were not included. In order to focus future activities, Chino decided to provide the NMED with estimates of the individual pathway calculations to illustrate and identify metals and pathways that drive risk and to provide additional information and context for them. Such an analysis is similar to a Baseline Risk Assessment because it involves a forward calculation of risk like the one approved for the Hurley Soils Investigation Unit (IU). In this case, however, it may be referred to as a screening level risk assessment since the HWCIU Remedial Investigation is still underway. It differs from the preliminary risk estimates provided by Neptune (2001) because it provides more detail as discussed above.

Exposure Point Concentrations

Exposure point concentrations (EPCs) for each constituent for each land feature type (i.e., overbanks, bars, and active channel) were obtained from Table 4 in the *Technical Memorandum* (Neptune, 2001). For this evaluation, Chino generated risk estimates for each scenario/feature using the median, geometric mean, and the 95 percent upper confidence limit (95 UCL) on the geometric mean for arsenic, copper, iron, manganese, and zinc. Lead was also considered and will be discussed separately below since it is evaluated based upon a different model. These EPCs are presented in Table 1. Neptune (2001) did not provide units for Table 4 in the

[1] Based on EPA guidance (EPA 1989), hazard quotients (HQs) are ratios of site specific dose based on exposure pathway for individual metals divided by the "safe dose". Hazard indices (HIs) are the sum of all HQs and do not reflect pathway or constituent contribution.

Technical Memorandum, therefore units of milligram per kilogram (mg/kg) were assumed based on the data as reported in the Phase I Remedial Investigation Report (Golder, 2000).

Non-Cancer and Cancer Dose Equations and Exposure Parameters

Chino calculated non-cancer and cancer risks using the risk assessment methodology presented in the *Technical Memorandum* (Neptune, 2001). HQs and cancer risk were calculated for each constituent for each individual pathway using equations that were obtained from Attachment 3, Equations for Calculation of Hazard Quotient and Cancer Risk (Neptune, 2001). The pathways included in each scenario were determined according to the table presented on the first page of Attachment 3 in the *Technical Memorandum*. For this exercise, only the On-Site Residential and the Off-Site Residential scenarios were evaluated because they yield the highest risk.

Because the usage of certain variables in the dose equations was not clear in the *Technical Memorandum* (Neptune, 2001), these variables had to be confirmed with Neptune. Correspondence with Neptune during 2001 resulted in minor changes to the equations. For example, while the variable $K_{f\text{-}s}$ included in the chicken and egg ingestion equations was renamed $F_{s;f}$, it had not been removed from the variable list. Following correspondence and clarification with Neptune (R. Perona, pers. comm., February 9, 2001), $K_{f\text{-}s}$ (and its definition) were removed from the equation. The corrected versions of the equations as used in Chino's analysis are presented in Attachment A with footnotes describing the changes.

Chino reconstructed HQs and cancer risk based on reasonable maximum exposure (RME). RME parameter estimates were obtained from the RME References table presented on page 25 and 26 of the *Technical Memorandum* (Neptune, 2001). Chino evaluated both adult and child receptors for the On-Site and Off-Site Residential scenarios. Neptune's parameter values that were used by Chino for evaluating these scenarios are presented in Table 2.

Particulate emission factor calculations and chemical-specific parameters, such as absorption fractions and transfer factors were also not included in the technical Memorandum (Neptune 2001) and were received directly from Neptune (R. Perona pers. comm., February 9, 2001). These values are presented in Tables 3 through 5.

Toxicity

Toxicity factors were also received directly from Neptune (R. Perona, pers. comm., February 9, 2001) since they were not included in the *Technical Memorandum*. These values are summarized in Table 6. Neptune obtained these values from the EPA's Integrated Risk

Information System (IRIS), the National Center for Environmental Assessment (NCEA), and previous risk assessments at the site conducted by Gradient. Reference concentrations (RfCs) and/or inhalation reference doses (RfDs) were not provided by Neptune for iron and zinc and, therefore, Chino did not evaluate the dust inhalation pathway for these two constituents.

Risk Characterization

Pathway-specific HQs and cancer risk calculated by Chino are presented in Table 7 and 8[2]. As discussed above, Chino is providing the risks by metal and by pathway to the NMED because this analysis provides a clearer picture of the issues at the HWCIU and is similar to how risks were estimated for Hurley Soils IU. In addition, if total non-cancer HIs are greater than 1 (as they are in Neptune's (2001) Table 7), USEPA guidance recommends that constituents be evaluated separately based upon similar endpoints and modes of toxicity. Copper, iron, manganese and zinc have different endpoints of toxicity and, therefore, they should be evaluated independently.

Table 7 shows that for the On-Site Residential Scenario, the HQs for individual constituents based on the 95 UCL on the geometric mean are below 1 except for iron, zinc and copper. HQs for iron were over 1 for the chicken ingestion and egg ingestion pathways (i.e., 6.2 and 4.4, respectively), while the zinc HQ for the chicken ingestion pathway slightly exceeds 1 (i.e., 1.7). The copper HQ for fruit and vegetable ingestion slightly exceeds 1 (i.e., 1.2).

With regard to iron non-cancer risk, the HQs are based on the 95 UCL on the geometric mean from Table 4 in the *Technical Memorandum* (i.e., 31,900 mg/kg) but this value is below the 95 UCL of the mean background (i.e., 39,335 mg/kg) as presented in the Technical Memorandum, Sediment Background Investigation (Golder, 2004). In addition, bioavailability was not included in the risk estimates. Iron at the IU appears to be present in the form of magnetite [Fe_3O_4], pyrite [FeS_2], chalcopyrite [$CuFeS_2$] and third-tier iron-bearing phases including silicates, hematite [Fe_2O_3], and ferricrete. Magnetite is ubiquitous and recalcitrant. It is highly resistant to weathering under natural conditions, and requires concentrated acid for dissolution. It is therefore very unlikely that any pathway that would require dissolution of magnetite (i.e., uptake into chickens or plants) could lead to exposure. Pyrite, chalcopyrite, hematite, and the various silicates are largely insoluble once the surface of the particles has oxidized. Mature ferricrete is resistant to chemical weathering as well.

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[2]Chino notes that although the equations were reproduced exactly as presented in the Technical Memorandum (2001) with consultations with Neptune, HQs for iron and zinc could not be reproduced precisely although our values were quite close (within one percent relative percent difference based upon our calculation of the RBSL values). As shown on Table 7, the parameters for fruit and vegetable ingestion, chicken ingestion, and egg ingestion do not differ between adults and children.

The solubility of iron is important with regard to the chicken and egg pathways because iron is absorbed by the chicken as well as the human who ingests meat or eggs. For example, Chino found research that indicated humans absorb heme iron at a higher rate than non-heme iron (Monsen and Balintfy, 1982). Non-heme iron has a bioavailability ranging from 2 percent in iron-replete individuals consuming a low availability meal to 20 percent in an iron deficient individual while heme iron ranged from 15 to 35 percent. Cooking degrades the highly bioavailable heme iron into the less-available non-heme iron by coagulation (Clark et al., 1997, Hazell, 1982). It is unknown whether the iron found in HWCIU would manifest as non-heme or heme iron in chicken tissue or eggs. Therefore, the consideration of bioavailability would significantly alter the perception of risk via this pathway.

In addition to the bioavailability of iron, the toxicity of iron needs further consideration as well. The Institute of Medicine has indicated that the Tolerable Upper Intake (UL) for iron in infants aged 0 to 12 months is 40 mg per day based on an endpoint of gastrointestinal effects (NAS 2003). The maximum detected iron concentration in E1 for all geomorphic features was 94,000 mg/kg which would equate to 19 mg iron for a child who incidentally ingested 200 mg soil entirely from that sample location. Site-related iron potentially present in chicken meat or eggs would be a fraction of this value. Since the iron RfD used in the *Technical Memorandum* is a provisional value cited in a "do not cite or quote" draft developed by USEPA, risk due to iron needs to be evaluated in light of the National Institute of Medicine Dietary Reference Intakes.

The HQ for zinc in the chicken ingestion pathway was 1.7. This is a slight exceedence even with the uncertainty surrounding the uptake factor. The Institute of Medicine has indicated that the UL for zinc in infants aged 0 to 12 months is 4-5 mg per day. The maximum detected zinc concentration in E1 for all geomorphic features was 5,160 mg/kg which would equate to 1 mg zinc for a child who incidentally ingested 200 mg soil entirely from that sample location. Site-related zinc potentially present in chicken meat would be a fraction of this value. This issue for toxicity must be reviewed for the chicken and egg ingestion pathways.

The HQ for copper in the ingestion of fruits/vegetables ingestion pathway was 1.2. This is a slight exceedence even with the uncertainty surrounding the plant uptake factor. While the plant and chicken/egg uptake factors for copper have been discussed as a source of uncertainty, Chino asserts that the reference dose used to derive the non-cancer risk deserves more scrutiny. As discussed for Hurley, given that the acute endpoint for copper toxicity was based on studies conducted with water and the most recent literature available from the Institute of Medicine (contained in Chino's comments on the Addendum to the Hurley Human Health Risk

Assessment) indicates that copper in food or supplements does not manifest the same acute response at the same levels as a bolus of water (NAS 2003), it does not appear that an HQ of 1.2 for the ingestion of fruits and vegetables pathway would require further evaluation. It appears that the focus for copper should be on how the toxicity criteria were derived and their application in the evaluation of these pathways, not refining the uptake factors.

Lead is not presented on the attached tables because it needs to be evaluated via a separate model, the integrated exposure uptake biokinetic model (IEUBK). However, the 95 UCL on the geometic mean concentrations for overbank, bars and active channel (i.e., 320 to 376 mg/kg) are less than USEPA's preliminary screening criteria of 400 mg/kg.

For the On-Site Residential Scenario, cancer risks represented by arsenic are greater than $1x10^{-6}$ for the soil ingestion, dermal contact, and fruit/vegetable, chicken and egg ingestion pathways, but all risks fall within EPA's acceptable risk range of $1x10^{-4}$ to $1x10^{-6}$ except the ingestion of chicken meat or eggs. In addition, the pathway contribution to overall risk for soil ingestion is 3 percent, dermal contact is 5 percent, and fruit/vegetable ingestion is 3 percent while chicken and egg ingestion make up the remaining 89 percent of the total cancer risk. These risks are summarized in Table 8.

In this regard, the mode of toxicity and bioavailability of arsenic needs to be discussed in more detail. For example, the slope factor for arsenic was based upon a cross-sectional study of 40,000 Taiwanese exposed to arsenic in drinking water which found significant excess skin cancer prevalence by comparison to 7,500 residents of Taiwan and Matsu who consumed relatively arsenic-free water (Tseng et al., 1968; Tseng, 1977). The bioavailability of arsenic in water differs from soil as it would also from soil to plants, chicken meat or eggs as evidenced by a study conducted by ARCO. In their pilot bioavailability study prepared by Battelle Laboratory (Johnson et al. 1989), arsenic in soil was administered to rabbits and bioavailability was on the order of 10 to 20 percent for ARCO's mining-related arsenic. Similarly, the arsenic at HWCIU largely occurs in sulfides and their weathering products and, when present in sulfides, arsenic is not bioavailable due to its insoluble nature. Weathering of sulfides may result in release of arsenic and subsequent precipitation with secondary minerals and/or adsorption. However, dissolved arsenic will co-precipitate with and adsorb onto iron (hydr)oxides. These factors need to be further evaluated with respect to the fact that 89 percent of the arsenic cancer risk is attributable to the chicken and egg ingestion pathways.

In addition, the maximum detected arsenic concentration in E1 for all geomorphic features was 15 mg/kg which would equate to 3 μg arsenic for a child who incidentally ingested

200 mg soil entirely from that sample location and arsenic was 100 percent solubilized. Site-related arsenic potentially present in chicken meat or eggs would be a fraction of this value. The Institute of Medicine did not establish an UL for arsenic but reports that the intakes of arsenic for all age groups ranged from 0.5 to 0.81 μg/kg/day (and converting 0.5 μg/kg/day for a 7.5 kg infant would be 3.7 μg) and the median intake by adult men and women were 2.0 to 2.9 μg/day and 1.7 to 2.1 μg/day, respectively. These aspects should be further evaluated with regard to arsenic exposure.

For the Off-Site Residential Scenario, which does not include chicken and egg ingestion, HQs and cancer risks are lower than for the On-Site Residential Scenario. These values are summarized in Tables 7 and 8. The only constituent that exceeds the non-cancer target value of 1 for any pathway is iron via soil ingestion by children and the HQ was 1.5. As discussed above, the mechanism for toxicity and the toxicity criteria proposed for evaluation must be considered further. The risks due to arsenic fall within EPA's acceptable risk range of 1×10^{-4} to 1×10^{-6}.

In addition to issues associated with toxicity and bioavailability discussed above, there is also considerable uncertainty associated with each of the three food ingestion pathways (as we have previously discussed during our meetings and conference calls over three years ago). These pathways (equations presented in Attachment A) rely upon conservative, literature-based estimates of biota transfer factors, moisture conversion factors, ingestion rates of dirt by chickens, and regional food consumption rates. The *Technical Memorandum* identified that chicken and egg pathways were driving 70 to 90 percent of the non-cancer and cancer risk, which is similar to the results provided herein, and cited metal uptake into meat and eggs as a major source of uncertainty. In addition, plant uptake factors were also noted as being a major source of uncertainty. However, Neptune (2001) also did not discuss other major sources of uncertainty for these exposure pathways such as the toxicity criteria or estimates of bioavailability.

Neptune (2001) recommended obtaining additional information to reduce uncertainty for the plant, chicken and egg ingestion pathways because of the wide range of variability associated with uptake factors citing in some cases a 150-fold difference between median and maximum uptake factors derived from the literature. However, maximum uptake factors are highly unlikely at this site simply due to the forms of metal present and the bioavailability of those forms in plants, chickens, or eggs as discussed above. In fact, for plants and birds, Dr. Lewis, MFG showed in the Ecological IU Risk Assessment that uptake of copper and other metals are directly related to the form of the metal present and pH. Based on the Ecological IU data, metal uptake into plants is lower than the median values cited by Neptune (2001). Nevertheless, based upon our analysis, we do not feel it is worth the time and expense to develop site-specific uptake factors for the following reasons:

- Copper and arsenic are the drivers for ingestion of fruit/vegetables. The copper HQ slightly exceeded one (i.e., 1.2) for this pathway and needs to be evaluated in light of its mode of toxicity. Arsenic risk via this pathway is one percent of the total arsenic risk and further refinement of the uptake factor will not impact the overall risk due to arsenic.

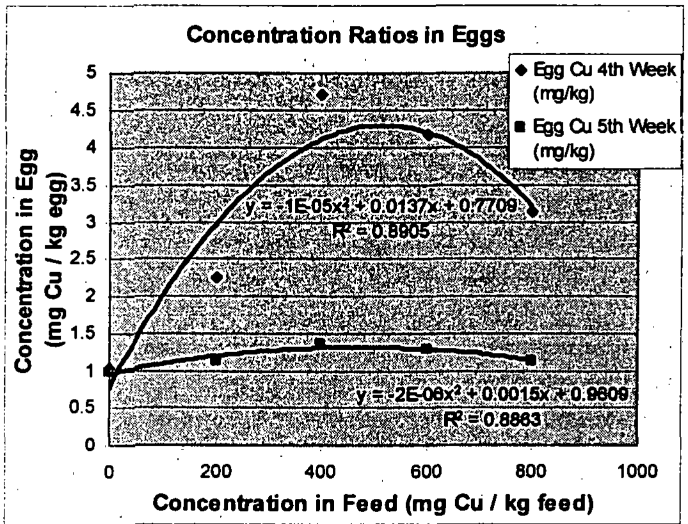
- Iron and arsenic are the drivers for the ingestion of chicken/eggs. Iron's non-cancer risk was based upon an exposure point concentration that is less than background concentrations. In addition, both iron and arsenic need to be re-evaluted based upon their respective toxicity criteria and bioavailability. The following factors relate specifically to a chicken/egg uptake study that make a cost effective study at Chino extremely difficult:

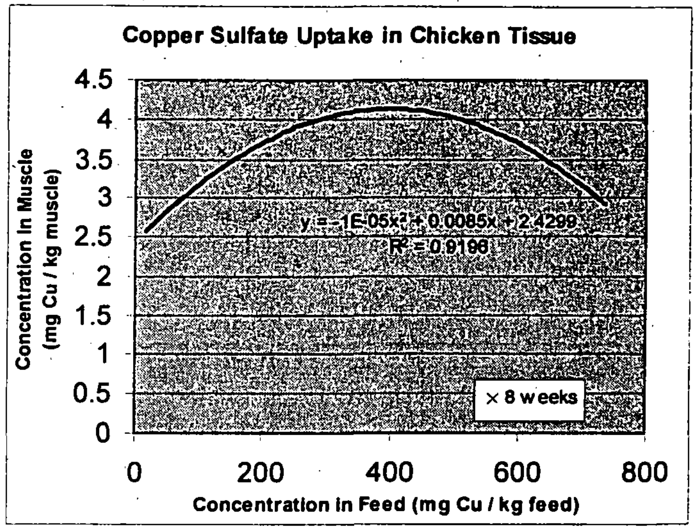
  Metal Interactions. There is evidence of interactions of the metals of interest upon dietary ingestion that may complicate an evaluation of the relationship between metal uptake and tissue concentrations. Palafox and Ho-a (1980) reported that 20 mg/g zinc in diet significantly decreased copper content of eggs collected 28 days after removal of zinc from the diet, and the authors cited three other studies (Johnson et al. 1962; Cox and Harris, 1962; and Hill, 1976) that reported zinc acting as a copper antagonist. Norvell et al. (1975) reported that dietary supplementation of 720 mg/kg copper did not affect tissue iron and liver zinc residues consistently, but did appear to lower muscle and kidney zinc levels in birds housed in individual cages. Chiou et al. (1998) reported that roxarsone, an organic arsenical, decreases liver copper content and can cause a decline in egg copper concentration.

  If soil/sediment concentrations with multiple metals are used for an uptake study, metal interactions may confound our ability to develop a predictive uptake model. This is especially true if multiple locations are chosen which have different ratios of metals, for example, at one area, copper may be 500 mg/kg and zinc 300 mg/kg versus another area where copper was 200 mg/kg and zinc was 600 mg/kg. In addition, each location would have a completely unique mix of metals (not just copper and zinc) that may influence each other in ways that could not be discernable unless a large scale research type study is conducted. However, if the focus is on just one location, then there is increased uncertainty relative to the fact that most of the metals of concern have quadratic uptake equations which level off at some threshold.

  Non-linear Uptake. The literature suggests that metal uptake rates in chickens for essential metals (Cu, Fe, Zn), which are homeostatically regulated, demonstrate non-linear uptake patterns in both eggs and meat. The National Research Council (1994) also classifies manganese as a required nutrient in chicken diets, and therefore, the uptake patterns of manganese in chickens are expected to approximate those of copper, iron, and zinc.

  An analysis of data from Chiou et al. (1998) and Norvell et al. (1975) suggests that copper uptake into meat and eggs is likely non-linear, specifically quadratic. In other words, the concentration in tissue (eggs or meat) increases as the concentration in feed increases (linear uptake) until a peak dietary level is reached, and then the concentration in tissue (eggs or meat) decreases as the concentration in feed continues to increase. The chickens in these studies were also fed *ad libitum*, and actual metal amounts ingested (mg/d) cannot be measured. The following graphs show the quadratic relationship that copper may have in eggs and meat. The equations of the curve are shown below them to demonstrate that the relationship between feed concentration and tissue concentration is more complex than a simple UF.





It is important to note in this analysis that at levels below 400 mg/kg in feed, the uptake rate appears linear. These graphs reflect controlled studies where there was only one metal being studied.

<u>Form of the Metal Administered</u>. The form of the metal used in the study can affect final tissue concentrations. For example, the data presented in Norvell et al. (1975) indicate that different metal salts are taken up at different rates. As multiple forms of the various metals are present in the soil at Chino, this issue may also confound our ability to develop a site-specific uptake model.

<u>Organ or tissue evaluated</u>. The concentrations of essential metals in muscle tissue remain relatively steady across deficient to sufficient diets, although other tissues' metal concentrations (e.g., liver, kidney, etc.) may vary. For example, iron, copper, and zinc concentrations in muscles remain relatively steady as intake varies compared to concentrations in organs such as the liver, spleen, kidneys, blood, and bone (Ng et al., 1982).

<u>Inter-bird variability</u>. The difference in individual bird metabolism can affect final tissue concentrations. Norvell et al. (1975) attribute their lack of precision in developing a relationship between metal ingested and tissue concentrations to inter-bird variability, among other factors.

<u>Bird housing</u>. The test birds' housing can affect metal uptake rates. Norvell et al. (1975) also attribute their lack of precision in results to bird housing and handling methods.

<u>Chicken age</u>. Metal uptake into tissue and eggs varies according to the age of the chickens. As demonstrated by the data presented in Norvell et al. (1975) and Chiou et al. (1997), metal uptake rates are highest in younger chickens.

<u>Egg production rates</u>. Egg production plays a key role in the homeostatic mechanisms of laying hens, as both a driver of metal uptake and a means of eliminating excess metals. For example, Morck and Austic (1981) demonstrate that hen iron absorption efficiency from an iron-deficient diet is higher when egg production rates are higher. Palafox and Ho-a (1980) observed a cessation of egg production in an excessively Zinc-supplemented diet.

<u>Feed/Soil uptake</u>. Chickens and ducks have been observed to adjust feed intake, and therefore potentially soil uptake, to control tissue stores of essential metals. For example, Morck and Austic (1981) observed increased feed intake in iron-deficient diets and diets meeting only the minimum iron requirements.

It will be extremely difficult to control for these variables given the complex array of metals and their distributions associated with the sediment in HWCIU. For these reasons, Chino believes that pursuing site-specific fruit/vegetable, chicken or egg uptake studies will produce data with additional uncertainty, will further delay the schedule, and will not resolve the key issues preventing us from making progress at this IU.

Path Forward

The main issue with HWCIU over the past five years has been how to adequately represent the constituent distributions (i.e., variability) in the various geomorphic features with regard to estimating exposure point concentrations. The issues with the risk-based screening levels (RBSLs) arose based upon NMED and Neptune's need for more data to describe the variability of constituent distributions in overbanks and other geomorphic features. The RBSLs were going to be a tool to use with XRF and confirmatory sample data in order to delineate areas of potential risk. The main objective of the *Technical Memorandum* was to focus Phase II efforts but the conclusions centered on refinement of the RBSLs via site-specific uptake studies. Since there are other issues that play into the sources of uncertainty such as solubility, bioavailability and toxicity, the revised *Technical Memorandum*, although anticipated to be more transparent and well documented, will not serve to resolve our ultimate issues, many of which have to do with adequately representing variability at the IU. Since it has taken over three years to obtain resolution of these RBSL's with no clear end, Chino has prepared this information for use by the NMED to make it clear that the RBSL's are not necessary for moving forward in this IU.

Chino is prepared to collect the necessary data in the form of XRF analysis with confirmatory samples to document constituent distribution in those areas that lack data. The

NMED's *Technical Memorandum* and this memo help frame the constituents, their distributions, and pathways that are driving risk. The additional data may be interpreted within the context and range of constituent concentrations represented by these two documents and the Phase I RI Report (Golder 2000). For example, there may be a concern that the upper end of the iron distribution has not been identified or that certain overbanks may have significantly different distributions of metals than documented in the Phase I RI Report. XRF data will fill in the gaps with respect to nature and extent of contamination. Chino does not believe it is necessary to do in-depth studies of garden, chickens or eggs to develop transfer factors in order to make progress on this IU. More study may be required to understand the chemical nature of iron or arsenic and how soluble their forms are for uptake. Chino has just completed the background characterization and these results will aid our future direction as well.

Conclusions

Chino has prepared this memorandum to assist NMED with moving forward at this IU. While it is anticipated that the revised *Technical Memorandum* will be more transparent and well documented, since the conclusions are the same, it will not help us move forward and make progress at this IU. Therefore, using the exact risk assessment methods presented in the *Technical Memorandum* (Neptune, 2001) and subsequent follow up conversations with Neptune, Chino has prepared a forward estimate of risk similar to what would be done if a Baseline Risk Assessment was completed for this site. This forward risk assessment serves to focus and prioritize which constituents by which pathway are an issue for further evaluation. Chino has identified several constituents and pathways that are potentially driving risk in the Hanover/Whitewater IU, specifically, iron and arsenic in the chicken and egg ingestion pathways.

Chino recommends NMED consider the relevancy of the chicken and egg ingestion pathways from the perspective of the toxicity criteria being used to evaluate them and the issues associated with bioavailability. Specifically, Chino requests that NMED not pursue the evaluation of these pathways.

In addition, Chino recommends NMED revisit the need to do site-specific plant uptake studies. The method used by Gradient (2000) in the Hurley Soils IU Baseline Risk Assessment may also be used for HWCIU. This method could have been incorporated into this analysis but we attempted to mirror Neptune (2001) precisely. However, if NMED requires the plant ingestion pathway to be evaluated, an analysis using Gradient's original methods which are based on an non-linear uptake model by Bechtel (1998) should be used. This method cannot be used in the derivation of RBSLs because it is an exponential equation that does not solve for one soil value that can be used as an RBSL.

Instead of site-specific uptake studies, further data collection in the form of XRF and confirmatory samples may answer unresolved questions with regard to nature and extent of contamination and, therefore, allow NMED to move forward. If the new data diverge significantly from those values submitted to NMED in the Phase I RI Report, then NMED may issue a Baseline Risk Assessment. Otherwise, if the data are consistent with those documented previously, we could move forward with the development of Remedial Action Criteria (RAC).

Chino appreciates the opportunity to submit these technical comments for your review. Please contact Mr. Robert Quintanar at (505) 537-4228 with any questions.

REFERENCES

Chiou, P.W., K.L. Chen, and B. Yu. 1997. Toxicity, tissue accumulation and residue in egg and excreta of copper in laying hens. Animal Feed Science Technology, 67: 49-60.

Chiou, P.W., K.L. Chen, and B. Yu. 1998. Effect of dietary organic arsenicals and cupric sulfate on copper toxicity, liver accumulation and residue in eggs and excreta of laying hens. Animal Feed Science Technology, 73: 161-171.

Cox, D.H. and Harris, D.L. 1962. Reduction of liver zanthine oxidase activity and iron storage protein in rats fed excess zinc. J. Nutr. 78:415-418 (as cited in Palafox, A. and Ho-A. E. 1980).

Golder and Associates (Golder). 2000. Phase I Remedial Investigation for Hanover and Whitewater Creeks Investigation Unit. Prepared for Chino Mines Company, Hurley, NM. May 25.

Golder. 2004. Technical Memorandum: Sediment Background Investigation. Prepared for Chino Mines Company, Hurley, NM. April 2.

Hill, C.H. 1976. Mineral interrelationships. Pages 281-300 in Trace elements in human health and disease. Anada S. Prasad, ed. Academic Press, New York, NY (as cited in Palafox, A. and Ho-A. E. 1980).

National Academy of Sciences (NAS). 2003. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. National Academy Press, Washington DC.

Johnson, D., Jr., Mehring, A.L., Savino, F.X. and Tritus, H.W. 1962. The tolerance of growing chickens for dietary zinc. Poultry Sci. 41:311-317 (as cited in Palafox, A. and Ho-A. E. 1980).

Johnson, J., Freeman, G.B. and Killinger, J.M. 1989. Pilot bioavailability study of lead and arsenic in soil following oral administration to rabbits. Prepared by Battelle Laboratory for ARCO Coal Company.

Monsen, E.R. and Balintfy, J.L. 1982. Calculating dietary iron bioavailability: Refinement and Computerization. J. of American Dietetic Association 80:307-311

Morck, T.A. and R.E. Austic. 1981. Iron requirements of White Leghorn Hens. Poultry Science, 60: 1497-1503.

Neptune and Company (Neptune). 2001. Technical Memorandum: Risk-Based Analysis of Phase I Data for the Hanover/Whitewater Investigation Unit.

Ng, Y.C., C.S. Colsher, and S.E. Thompson. 1982. Transfer coefficients for assessing the dose from radionuclides in meat and eggs. Lawrence Livermore National Laboratory. NUREG/CR-2976.

Palafox, A. and Ho-A. E. 1980. Effects of zinc toxicity in laying White Leghorn Pullets and Hens. Poultry Science, 59: 2024-2028.

Tseng, W.P. 1977. Effects and dose-response relationships of skin cancer and blackfoot disease with arsenic. Environ. Health Perspect. 19: 109-119.

Tseng, W.P., H.M. Chu, S.W. How, J.M. Fong, C.S. Lin and S. Yeh. 1968. Prevalence of skin cancer in an endemic area of chronic arsenicism in Taiwan. J. Natl. Can0cer. Inst. 40(3): 453-463.

U.S. Environmental Protection Agency (EPA). 1989. Risk Assessment Guidance for Superfund.

Table 1. Exposure Point Concentrations (obtained from Table 4 [Neptune, 2001])

Constituent	95 UCL (mg/kg)	Geometric Mean (mg/kg)	Median (mg/kg)
Overbanks			
Arsenic	9.39	5.27	3.82
Copper	1,610	1,110	938
Iron	31,900	27,400	23,600
Manganese	1,350	1,230	1,000
Zinc	1,960	1,140	865
Bars			
Arsenic	12.3	9.13	8.15
Copper	946	787	677
Iron	61,200	55,800	50,600
Manganese	1,640	1,240	918
Zinc	1,580	1,090	720
Channel			
Arsenic	13.3	9.11	7.8
Copper	699	628	612
Iron	55,300	46,700	45,600
Manganese	1,540	1,260	1,120
Zinc	1,760	1,300	1,050

Table 2. RME Exposure Parameters (obtained from RME References Table [Neptune, 2001; p. 25-26])

	Symbol	units	On-Site Residents		Off-Site Residents	
			Child	Adult	Child	Adult
General Factors						
Averaging Time (cancer)	AT_{carc}	day	--	27375	--	27375
Averaging Time (noncancer)	AT_{nc}	day	2190	10950	2190	10950
Body Weight	BW	kg	17.4	71.8	17.4	71.8
Exposure Frequency	EF	day/yr	350	350	350	350
Exposure Duration (cancer)	ED_c	year	--	30	--	30
Exposure Duration (noncancer)	ED_{nc}	year	6	24	6	24
Soil - Ingestion (Oral)						
Incidental Soil Ingestion Rate	IR_s	mg/day	200	50	200	50
Outdoor Activity Soil Loading Enrichment Factor	E_{out}	unitless	10	10	10	10
Time Onsite Spent Outdoors	$T_{site,out}$	hr/day	3	3	3	3
Time Onsite Spent Indoors	$T_{site,\ in}$	hr/day	19.4	16	19.4	16
Total Time Spent Outdoors	T_{out}	hr/day	4.6	3	4.6	3
Total Time Spent Indoors	T_{in}	hr/day	19.4	21	19.4	21
Time Spent Sleeping or Napping	T_{sleep}	hr/day	11.5	8	11.5	8
Site-Specific Soil Ingestion Rate	$IR_{s,site}$	mg/day	137	50	137	50
Soil - Inhalation of Dust						
Inhalation Rate	InhR	m^3/hr	0.42	0.63	0.42	0.63
Exposure Time	ET	hr/day	22.4	19	22.4	19
Soil - Dermal Contact						
Surface Area of Hand	SA_h	cm^2	420	840	420	840
Surface Area of Arm	SA_a	cm^2	1020	1140	1020	1140
Surface Area of Leg	SA_l	cm^2	2030	2070	2030	2070
Soil Adherence Factor of Hand	AF_h	mg/cm^2-event	0.25	0.18	0.25	0.18
Soil Adherence Factor of Arm	AF_a	mg/cm^2-event	0.059	0.054	0.059	0.054
Soil Adherence Factor of Leg	AF_l	mg/cm^2-event	0.059	0.022	0.059	0.022
Surface-Area-Adjusted Adherence Factor	AF_{adj}	mg/event	284.95	258.3	284.95	258.3
Exposure Frequency for Dermal Contact with Soil	EF_{derm}	event/yr	350	350	350	350
Ingestion of Garden Produce and Fruit						
Ingestion Rate of Home-Grown Vegetables	IR_v	g/kg-day	1.2	1.2	1.2	1.2
Ingestion Rate of Home-Grown Fruits	IR_f	g/kg-day	1.4	1.4	1.4	1.4
Exposure Frequency for Ingesting Food Products	EF_{food}	day/yr	365	365	365	365
Depth of Contaminated Zone	$depth_{cz}$	cm	0.15	0.15	0.15	0.15
Effective Rooting Depth of Garden Vegetables	$depth\_root_v$	cm	0.3	0.3	0.45	0.45
Effective Rooting Depth of Fruit Trees	$depth\_root_f$	cm	1	1	1	1
Conversion Factor for Fruits	CvF_p	kg ww/kg dw	0.15	0.15	0.15	0.15
Ingestion of Chicken						
Chicken Ingestion Rate	IR_c	kg/year	1.3	1.3	--	--
Exposure Frequency for Ingesting Food Products	EF_{food}	day/yr	365	365	--	--
Uptake Rate of Dry Feed	UR_f	kg/day	0.1	0.1	--	--
Fraction of Soil in Chicken Feed	$F_{s,f}$	unitless	0.3	0.3	--	--
Ingestion of Eggs						
Egg Ingestion Rate	IR_e	kg/year	1.05	1.05	--	--
Exposure Frequency for Ingesting Food Products	EF_{food}	day/yr	365	365	--	--
Uptake Rate of Dry Feed	UR_f	kg/day	0.1	0.1	--	--
Fraction of Soil in Chicken Feed	$F_{s,f}$	unitless	0.3	0.3	--	--

Table 3. Absorption Fractions (R. Perona, pers. comm., February 9, 2001)

Constituent	Dermal Absorption Fraction (unitless)	Oral Absorption Fraction (unitless)
Arsenic	0.03	0.05
Copper	0.01	1
Iron	0.01	0.05
Manganese	0.01	0.05
Zinc	0.01	0.05

Table 4. Transfer Factors (R. Perona, pers. comm., February 9, 2001)

Constituent	Baes Br Value (unitless)	Plant/Soil Concentration Ratio (unitless)	Chicken Transfer Factor (mg/kg meat per mg/d)	Egg Transfer Factor (mg/kg egg per mg/d)
Arsenic	6.0E-03	9.0E-04	8.3E-01	8.0E-01
Copper	2.5E-01	3.8E-02	5.1E-01	4.9E-01
Iron	1.0E-03	1.5E-04	1.5E+00	1.3E+00
Manganese	5.0E-02	7.5E-03	5.0E-02	6.5E-02
Zinc	9.0E-01	1.4E-01	6.5E+00	2.6E+00

Table 5. Particulate Emission Factor and Input Parameters (R. Perona, pers. comm., February 9, 2001)

Parameter	Value	Units	Description
Q/C	35.1	g/m^2-s per kg/m^3	inverse of mean conc for 30 acre source (high-end estimate in EPA 1996)
V	0.1	unitless	fraction of vegetative cover (Best Professional Judgment for reasonable worst case conditions)
Um	3.9	m/s	mean annual wind speed (from Gradient/IT 1998)
Z	7	m	height above surface
Z0	0.05	m	surface roughness height (Cowherd 1985: Fig. 3-6, higher-end estimate for grassland)
Ut	0.625	m/s	threshold friction velocity (EPA 1996: default for App D., eq. 4)
Ut7	7.7	m/s	equivalent threshold wind speed at 7 m (EPA 1996: calculated using App. D, eq. 4)
F(x)	0.6	unitless	function dependent on Um/Ut-7; 'best-fit' equations using Math Cad software
PEF	4.96E+07	m^3/kg	particulate emission factor

Table 6. Toxicity Factors (R. Perona, pers.comm., February 9, 2001)

Constituent	Oral RfD (mg/kg-d)	Source	Inhal RfD (mg/kg-d)	Source	Oral SF $(mg/kg\text{-}d)^{-1}$	Source	Inhal SF $(mg/kg\text{-}d)^{-1}$	Source
Arsenic	3.0E-04	IRIS			1.5E+00	IRIS	1.5E+01	IRIS
Copper	4.0E-02	NCEA	4.4E-03	Gradient				
Iron	3.0E-01	NCEA						
Manganese	4.7E-02	IRIS	1.4E-05	IRIS				
Zinc	3.0E-01	IRIS						

IRIS: EPA's Intergrated Risk Information System, www.epa.gov/iris/
NCEA: EPA's National Center for Environmental Assessment
Gradient: Gradient Risk Assessment

Table 7. Summary of Non-Carcinogenic Risk Estimates by Exposure Scenario and Land Feature EPC

Scenario	Soil Concentration	Calculated HI (adult)	Calculated HI (child)	Soil Ingestion (adult)	Soil Ingestion (child)	Dust Inhalation (adult)	Dust Inhalation (child)	Soil Dermal (adult)	Soil Dermal (child)	Fruit & Veg Ingestion	Chicken Ingestion	Egg Ingestion
Non-cancer Risk for On-Site Residential (Overbanks)												
RME HQs - **EPCs based on 95% UCL on geometric mean (LogMean UCL from Neptune's Table 4 of January 24, 2001 Tech Memo)												
Copper	1,610	2.7	3.0	0.027	0.3	0.001	0.004	0.001	0.006	1.2	0.8	0.6
Iron (a)	31,900	11	12	0.07	0.8	--	--	0.07	0.33	0.01	6.2	4.4
Manganese	1,350	0.64	1.6	0.02	0.2	0.31	1.01	0.02	0.09	0.17	0.06	0.06
Zinc (a)	1,960	2.9	3.0	0.004	0.05	--	--	0.005	0.02	0.71	1.7	0.54
RME HQs - **EPCs based on geometric mean (LogMean from Neptune's Table 4 of January 24, 2001 Tech Memo)												
Copper	1,110	1.8	2.0	0.019	0.2	0.001	0.003	0.001	0.004	0.8	0.6	0.4
Iron (a)	27,400	9.2	10	0.06	0.7	--	--	0.06	0.29	0.01	5.3	3.7
Manganese	1,230	0.58	1.5	0.02	0.2	0.28	0.92	0.02	0.08	0.2	0.05	0.05
Zinc (a)	1,140	1.7	1.7	0.003	0.03	--	--	0.00	0.01	0.4	1.0	0.3
RME HQs - **EPCs based on median from Neptune's Table 4 of January 24, 2001 Tech Memo												
Copper	938	1.6	1.7	0.02	0.2	0.001	0.002	0.001	0.004	0.7	0.5	0.4
Iron (a)	23,600	7.9	8.7	0.05	0.6	--	--	0.05	0.2	0.01	4.6	3.2
Manganese	1,000	0.47	1.2	0.01	0.2	0.2	0.7	0.01	0.07	0.1	0.04	0.04
Zinc (a)	865	1.3	1.3	0.00	0.02	--	--	0.00	0.01	0.3	0.7	0.2
Non-cancer Risk for Off-Site Residential (Borrow Fill - Bars)												
RME HQs - **EPCs based on 95% UCL on geometric mean (LogMean UCL from Neptune's Table 4 of January 24, 2001 Tech Memo)												
Copper	946	0.6	0.7	0.02	0.2	0.001	0.00	0.001	0.00	0.5	--	--
Iron (a)	61,200	0.3	2.2	0.1	1.5	--	--	0.1	0.6	0.02	--	--
Manganese	1,640	0.6	1.8	0.02	0.3	0.4	1.2	0.02	0.1	0.2	--	--
Zinc (a)	1,580	0.4	0.5	0.004	0.04	--	--	0.004	0.02	0.4	--	--
RME HQs - **EPCs based on geometric mean (LogMean from Neptune's Table 4 of January 24, 2001 Tech Memo)												
Copper	787	0.46	0.60	0.01	0.15	0.001	0.002	0.001	0.003	0.5	--	--
Iron (a)	55,800	0.3	2.0	0.1	1.4	--	--	0.1	0.6	0.02	--	--
Manganese	1,240	0.4	1.3	0.02	0.2	0.3	0.9	0.02	0.1	0.1	--	--
Zinc (a)	1,090	0.3	0.3	0.002	0.0	--	--	0.003	0.01	0.3	--	--
RME HQs - **EPCs based on median from Neptune's Table 4 of January 24, 2001 Tech Memo												
Copper	677	0.4	0.5	0.01	0.1	0.0005	0.002	0.0006	0.003	0.4	--	--
Iron (a)	50,600	0.2	1.8	0.1	1.3	--	--	0.1	0.5	0.02	--	--
Manganese	918	0.3	1.0	0.01	0.1	0.2	0.7	0.01	0.1	0.1	--	--
Zinc (a)	720	0.2	0.2	0.002	0.02	--	--	0.002	0.01	0.2	--	--

Table 7. Summary of Non-Carcinogenic Risk Estimates by Exposure Scenario and Land Feature EPC

Scenario	Soil Concentrat ion	Calculated HI (adult)	Calculated HI (child)	Soil Ingestion (adult)	Soil Ingestion (child)	Dust Inhalation (adult)	Dust Inhalation (child)	Soil Dermal (adult)	Soil Dermal (child)	Fruit & Veg Ingestion	Chicken Ingestion	Egg Ingestion
Non-cancer Risk for Off-Site Residential (Borrow Fill - Active Channel)												
RME HQs - **EPCs based on 95% UCL on geometric mean (LogMean UCL from Neptune's Table 4 of January 24, 2001 Tech Memo)												
Copper	699	0.4	0.5	0.01	0.1	0.001	0.002	0.001	0.003	0.4	--	--
Iron (a)	55,300	0.3	2.0	0.1	1.4	--	--	0.1	0.6	0.02	--	--
Manganese	1,540	0.5	1.6	0.02	0.2	0.4	1.1	0.02	0.1	0.1	--	--
Zinc (a)	1,760	0.5	0.5	0.004	0.04	--	--	0.004	0.02	0.5	--	--
RME HQs - **EPCs based on geometric mean (LogMean from Neptune's Table 4 of January 24, 2001 Tech Memo)												
Copper	628	0.37	0.48	0.01	0.12	0.0005	0.001	0.001	0.002	0.4	--	--
Iron (a)	46,700	0.2	1.7	0.1	1.2	--	--	0.1	0.5	0.01	--	--
Manganese	1,260	0.4	1.3	0.02	0.2	0.3	0.9	0.02	0.1	0.1	--	--
Zinc (a)	1,300	0.4	0.4	0.00	0.03	--	--	0.003	0.01	0.4	--	--
RME HQs - **EPCs based on median from Neptune's Table 4 of January 24, 2001 Tech Memo												
Copper	612	0.4	0.5	0.01	0.1	0.0004	0.001	0.001	0.002	0.3	--	--
Iron (a)	45,600	0.2	1.6	0.1	1.1	--	--	0.1	0.5	0.01	--	--
Manganese	1,120	0.4	1.2	0.02	0.2	0.3	0.8	0.02	0.1	0.1	--	--
Zinc (a)	1,050	0.3	0.3	0.002	0.03	--	--	0.002	0.01	0.3	--	--

(a) there is no inhalation RfC for iron and zinc.

Table 8. Summary of Carcinogenic Risk Estimates by Exposure Scenario and Land Feature EPC

Scenario	Soil Concentration	Calculated ICR (adult+child)	Soil Ingestion (adult+child)	Dust Inhalation (adult+child)	Soil Dermal (adult+child)	Fruit & Veg Ingestion	Chicken Ingestion	Egg Ingestion
Carcinogenic Risk for On-Site Residential (Overbanks)								
RME ICRs - **EPCs based on 95% UCL on geometric mean (LogMean UCL from Neptune's Table 4 of January 24, 2001 Tech Memo)								
Arsenic	9.39	3.6E-04	1.2E-05	2.4E-07	2.0E-05	4.1E-06	1.8E-04	1.4E-04
RME ICRs - **EPCs based on geometric mean (LogMean from Neptune's Table 4 of January 24, 2001 Tech Memo)								
Arsenic	5.27	2.0E-04	6.5E-06	1.4E-07	1.1E-05	2.3E-06	1.0E-04	8.0E-05
RME ICRs - **EPCs based on median from Neptune's Table 4 of January 24, 2001 Tech Memo								
Arsenic	3.82	1.5E-04	4.7E-06	1.0E-07	8.1E-06	1.7E-06	7.4E-05	5.8E-05
Carcinogenic Risk for Off-Site Residential (Borrow Fill - Bars)								
RME ICRs - **EPCs based on 95% UCL on geometric mean (LogMean UCL from Neptune's Table 4 of January 24, 2001 Tech Memo)								
Arsenic	12.3	4.6E-05	1.5E-05	3.2E-07	2.6E-05	4.1E-06	--	--
RME ICRs - **EPCs based on geometric mean (LogMean from Neptune's Table 4 of January 24, 2001 Tech Memo)								
Arsenic	9.13	3.4.E-05	1.1.E-05	2.4.E-07	1.9.E-05	3.0.E-06	--	--
RME ICRs - **EPCs based on median from Neptune's Table 4 of January 24, 2001 Tech Memo								
Arsenic	8.15	3.0E-05	1.0E-05	2.1E-07	1.7E-05	2.7E-06	--	--
Carcinogenic Risk for Off-Site Residential (Borrow Fill - Active Channel)								
RME ICRs - **EPCs based on 95% UCL on geometric mean (LogMean UCL from Neptune's Table 4 of January 24, 2001 Tech Memo)								
Arsenic	13.3	4.9E-05	1.6E-05	3.5E-07	2.8E-05	4.4E-06	--	--
RME ICRs - **EPCs based on geometric mean (LogMean from Neptune's Table 4 of January 24, 2001 Tech Memo)								
Arsenic	9.11	3.4E-05	1.1E-05	2.4E-07	1.9E-05	3.0E-06	--	--
RME ICRs - **EPCs based on median from Neptune's Table 4 of January 24, 2001 Tech Memo								
Arsenic	7.80	2.9E-05	9.6E-06	2.0E-07	1.7E-05	2.6E-06	--	--

Attachment A
Hazard and Risk Equations

Hazard Index

$$HI_{on} = HQ_{soil ing} + HQ_{dust inh} + HQ_{dermal} + HQ_{fruit \& veg ing} + HQ_{chicken ing} + HQ_{egg ing}$$

HI_{on}	=	Hazard Index for the On-site Residential Receptor (unitless)
$HQ_{soil\ ing}$	=	Hazard Quotient for the Soil Ingestion Pathway (unitless)
$HQ_{dust\ inh}$	=	Hazard Quotient for the Dust Inhalation Pathway (unitless)
HQ_{dermal}	=	Hazard Quotient for the Dermal Contact Pathway (unitless)
$HQ_{fruit\ \&\ veg\ ing}$	=	Hazard Quotient for the Garden Fruit and Vegetable Ingestion Pathway (unitless)
$HQ_{chicken\ ing}$	=	Hazard Quotient for the Chicken Ingestion Pathway (unitless)
$HQ_{egg\ ing}$	=	Hazard Quotient for the Egg Ingestion Pathway (unitless)

$$HI_{off} = HQ_{soil ing} + HQ_{dust inh} + HQ_{dermal} + HQ_{fruit \& veg ing}$$

HI_{off}	=	Hazard Index for the Off-site Residential Receptor (unitless)
$HQ_{soil\ ing}$	=	Hazard Quotient for the Soil Ingestion Pathway (unitless)
$HQ_{dust\ inh}$	=	Hazard Quotient for the Dust Inhalation Pathway (unitless)
HQ_{dermal}	=	Hazard Quotient for the Dermal Contact Pathway (unitless)
$HQ_{fruit\ \&\ veg\ ing}$	=	Hazard Quotient for the Garden Fruit and Vegetable Ingestion Pathway (unitless)

Hazard Quotient - Incidental Soil Ingestion

$$HQ_{\text{soiling}} = \frac{\left(C_{s,i} \times IR_{s,site} \times EF \times ED_{nc} \times 10^{-6}\, kg/mg\right)}{\left(BW \times AT_{nc} \times RfD_{ing,i}\right)}$$

$HQ_{soil\ ing}$	=	Hazard Quotient for the Soil Ingestion Pathway (unitless)
$C_{s,i}$	=	Concentration of Contaminant *i* in Exposure Area Soil (mg/kg soil)
$IR_{s,site}$	=	Site-Specific Soil Ingestion Rate (mg/d)
EF	=	Exposure Frequency [d/yr]
ED_{nc}	=	Exposure Duration (noncancer) [yr]
BW	=	Body Weight (kg)
AT_{nc}	=	Averaging Time (noncancer) [d]
$RfD_{ing,i}$	=	Ingestion Reference Dose, contaminant *i* (mg/kg-d)

$$IR_{s,site} = IR_s \times \left\{ \frac{\left(E_{out} \times T_{site,out}\right) + \left(\dfrac{T_{site,in}}{E_{out}}\right)}{\left(E_{out} \times T_{out}\right) + \left[\dfrac{\left(T_{in} - T_{sleep}\right)}{E_{out}}\right]} \right\}$$

$IR_{s,site}$	=	Site-Specific Soil Ingestion Rate (mg/d)
IR_s	=	Incidental Soil Ingestion Rate (mg/d)
E_{out}	=	Outdoor Activity Soil Loading Enrichment Factor (unitless)
$T_{site,out}$	=	Time Onsite Spent Outdoors (hr/d)
$T_{site,\ in}$	=	Time Onsite Spent Indoors (hr/d)
T_{out}	=	Total Time Spent Outdoors (hr/d)
T_{in}	=	Total Time Spent Indoors (hr/d)
T_{sleep}	=	Time Spent Sleeping or Napping (hr/d)

Hazard Quotient - Dust Inhalation

$$HQ_{\text{dust inh}} = \frac{\left(C_{s,i} \times InhR \times ET \times EF \times ED_{nc}\right)}{\left(PEF \times BW \times AT_{nc} \times RfD_{inh,i}\right)}$$

$HQ_{\text{dust inh}}$	=	Hazard Quotient for the Dust Inhalation Pathway (unitless)
$C_{s,i}$	=	Concentration of Contaminant *i* in Exposure Area Soil (mg/kg soil)
InhR	=	Inhalation Rate (m^3/hr)
ET	=	Exposure Time (hr/d)
EF	=	Exposure Frequency (d/yr)
ED_{nc}	=	Exposure Duration (noncancer) [yr]
PEF	=	Particulate Emission Factor (m^3/kg)
BW	=	Body Weight (kg)
AT_{nc}	=	Averaging Time (noncancer) [d]
$RfD_{inh,i}$	=	Inhalation Reference Dose, contaminant *i* (mg/kg-d)

$$PEF = \frac{\left(Q/C \times 3600\,\text{sec}/hr\right)}{\left(0.036 \times (1-v) \times \left(\frac{U_m}{U_{t-7}}\right)^3 \times F(x)\right)}$$

PEF	=	Particulate Emission Factor (m^3/kg)
Q/C	=	Inverse of Mean Concentration at the Center of a Square Source Area (g/m^2-sec per kg/m^3)
v	=	Fraction of Vegetative Cover (unitless)
U_m	=	Mean Annual Windspeed (m/sec)
U_{t-7}	=	Equivalent Threshold Wind Speed at 7 m (m/s)
F(x)	=	Function dependent on U_m/U_{t-7}

$$U_{t-7} = \left(\frac{U_t}{0.4}\right) \times \ln\left(\frac{z}{z_0}\right)$$

U_{t-7}	=	Equivalent Threshold Wind Speed at 7 m (m/s)
U_t	=	Threshold Friction Velocity (m/s)
z	=	Height Above Surface (m)
z_0	=	Surface Roughness Height (m)

Hazard Quotient - Dermal Contact

$$HQ_{\text{dermal}} = \frac{\left(C_{s,i} \times ABS_i \times AF_{adj} \times EF_{derm} \times ED_{nc} \times 10^{-6}\,kg/mg\right)}{\left(BW \times AT_{nc} \times RfD_{ing,i} \times F_{oral,i}\right)}$$

HQ_{dermal}	=	Hazard Quotient for the Dermal Contact Pathway (unitless)
$C_{s,i}$	=	Concentration of Contaminant *i* in Exposure Area Soil (mg/kg soil)
ABS_i	=	Dermal Absorption Factor, contaminant *i* (unitless)
AF_{adj}	=	Surface-Area-Adjusted Adherence Factor (mg/event)
EF_{derm}	=	Exposure Frequency for Dermal Contact with Soil (event/yr)
ED_{nc}	=	Exposure Duration (noncancer) [yr]
BW	=	Body Weight (kg)
AT_{nc}	=	Averaging Time (noncancer) [d]
$RfD_{ing,i}$	=	Ingestion Reference Dose, contaminant *i* (mg/kg-d)
$F_{oral,i}$	=	Oral Absorption Factor for Contaminant *i* (unitless)

$$AF_{adj} = \left(SA_h \times AF_h\right) + \left(SA_a \times AF_a\right) + \left(SA_l \times AF_l\right)$$

AF_{adj}	=	Surface-Area-Adjusted Adherence Factor (mg/event)
SA_h	=	Surface Area of Hand (cm^2)
SA_a	=	Surface Area of Arm (cm^2)
SA_l	=	Surface Area of Leg (cm^2)
AF_h	=	Soil Adherence Factor of Hand (mg/cm^2-event)
AF_a	=	Soil Adherence Factor of Arm (mg/cm^2-event)
AF_l	=	Soil Adherence Factor of Leg (mg/cm^2-event)

Hazard Quotient - Fruit and Vegetable Ingestion

$$HQ_{\text{fruit \& veg ing}} = \frac{\left(C_{s,i} \times K_{p-s,i} \times \left[\left(IR_v \times \frac{depth_{cz}}{depth\_root_v}\right) + \left(IR_f \times \frac{depth_{cz}}{depth\_root_f}\right)\right] \times EF_{food} \times ED_{nc} \times 10^{-3}\, kg/g\right)}{\left(AT_{nc} \times RfD_{ing,i}\right)}$$

$HQ_{\text{fruit \& veg ing}}$	=	Hazard Quotient for the Garden Fruit and Vegetable Ingestion Pathway (unitless)
$C_{s,i}$	=	Concentration of Contaminant *i* in Exposure Area Soil (mg/kg soil)
$K_{p\text{-}s,\,i}$	=	plant:soil concentration ratio, contaminant I (mg/kg wet weight plant per mg/kg soil)*
IR_v	=	Ingestion Rate of Home-Grown Vegetables (g/kg-d)
IR_f	=	Ingestion Rate of Home-Grown Fruits (g/kg-d)
$depth_{cz}$	=	Depth of Contaminated Zone (cm)
$depth\_root_v$	=	Effective Rooting Depth of Garden Vegetables (cm)
$depth\_root_f$	=	Effective Rooting Depth of Fruit Trees (cm)
EF_{food}	=	Exposure Frequency for Ingesting Food Products (d/yr)
ED_{nc}	=	Exposure Duration (noncancer) [yr]
AT_{nc}	=	Averaging Time (noncancer) [d]
$RfD_{ing,i}$	=	Ingestion Reference Dose, contaminant *i* (mg/kg-d)

$$K_{p-s,i} = B_r \times CvF_p$$

$K_{p\text{-}s,\,i}$	=	plant:soil concentration ratio, contaminant I (mg/kg wet weight plant per mg/kg soil)*
B_r	=	Baes Soil-to-Root Transfer Factor (mg/kg dry weight plant per mg/kg soil)
CvF_p	=	Conversion Factor for Fruits (unitless)

* The fruit and vegetable ingestion equation presented in the 2001 HHRA memo has an error. Kp-s is represented as mg/kg dry weight plant per mg/kg soil, and is multiplied by the conversion factor used to convert from dry to wet weight. However, in spreadsheets provided to Chino on February 9, 2001, (R. Perona, pers. comm) Kp-s is defined as the Baes transfer factor (mg/kg dry weight) multiplied by the conversion factor used to convert from dry to wet weight. Therefore, the equation appears to include the conversion factor twice. This equation presents the corrected algorithm.

** A single equation was used to evaluate both adults and children. Because Neptue assigned adults and children the same fruit ingestion rate, vegetable ingestion rate, and exposure freqency, the only variables that differ for adults and children are the exposure duration and averaging time. For noncancer hazard evaluations, these exposure parameters cancel each other out. As a result, the hazard quotients for this pathway are identical for the adult and child receptors.

Hazard Quotient - Chicken Ingestion

$$HQ_{\text{chicken ing}} = \frac{\left(C_{s,i} \times TF_{s-c,i} \times UR_f \times F_{s-f} \times IR_c \times EF_{food} \times ED_{nc} \times 10^{-3}\,kg/g\right)}{\left(AT_{nc} \times RfD_{ing,i}\right)}$$

$HQ_{\text{chicken ing}}$	=	Hazard Quotient for the Chicken Ingestion Pathway (unitless)
$C_{s,i}$	=	Concentration of Contaminant *i* in Exposure Area Soil (mg/kg soil)
$TF_{s\text{-}c,i}$	=	Soil-Chicken Transfer Factor, contaminant *i* (mg/kg chicken per mg/d)
UR_f	=	Uptake Rate of Dry Feed (kg/d)
$F_{s,f}$	=	Fraction of Soil in Chicken Feed (unitless)
IR_c	=	Chicken Ingestion Rate (g/kg-d)
EF_{food}	=	Exposure Frequency for Ingesting Food Products (d/yr)
ED_{nc}	=	Exposure Duration (noncancer) [yr]
AT_{nc}	=	Averaging Time (noncancer) [d]
$RfD_{ing,i}$	=	Ingestion Reference Dose, contaminant *i* (mg/kg-d)

* The chicken ingestion equation presented in the 2001 HHRA memo has an error. Neptune (R. Perona, pers. comm) confirmed that Kf-s was renamed Fs,f, and Kf-s (and its definition) should be deleted from the equation's list of parameters. The corrected version of the equation is shown here.

** The units shown in the 2001 HHRA memo for the chicken ingestion rate (kg/yr) appear to be in error. A dimensional analysis shows that the units for this parameter presented in the RME references table of the memo (g/kg-d) are mathematically correct. These units have been assumed, and are presented here.

Hazard Quotient - Egg Ingestion

$$HQ_{\text{egging}} = \frac{\left(C_{s,i} \times TF_{s-e,i} \times UR_f \times F_{s-f} \times IR_e \times EF_{food} \times ED_{nc} \times 10^{-3}\,kg/g\right)}{\left(AT_{nc} \times RfD_{ing,i}\right)}$$

$HQ_{egg\ ing}$	=	Hazard Quotient for the Egg Ingestion Pathway (unitless)
$C_{s,i}$	=	Concentration of Contaminant *i* in Exposure Area Soil (mg/kg soil)
$TF_{s\text{-}e,i}$	=	Soil-Egg Transfer Factor, contaminant I (mg/kg egg per mg/d)
UR_f	=	Uptake Rate of Dry Feed (kg/d)
$F_{s,f}$	=	Fraction of Soil in Chicken Feed (unitless)
IR_e	=	Egg Ingestion Rate (g/kg-d)
EF_{food}	=	Exposure Frequency for Ingesting Food Products (d/yr)
ED_{nc}	=	Exposure Duration (noncancer) [yr]
AT_{nc}	=	Averaging Time (noncancer) [d]
$RfD_{ing,i}$	=	Ingestion Reference Dose, contaminant *i* (mg/kg-d)

* The egg ingestion equation presented in the 2001 HHRA memo has an error. Neptune (R. Perona, pers. comm) confirmed that Kf-s was renamed Fs,f, and Kf-s (and its definition) should be deleted from the equation's list of parameters. The corrected version of the equation is shown here.

** The units shown in the 2001 HHRA memo for the egg ingestion rate (kg/yr) appear to be in error. A dimensional analysis shows that the units for this parameter presented in the RME references table of the memo (g/kg-d) are mathematically correct. These units have been assumed, and are presented here.

Cumulative Risk

$$ICR_{on} = ICR_{\text{soil ing}} + ICR_{\text{dust inh}} + ICR_{\text{dermal}} + ICR_{\text{fruit \& veg ing}} + ICR_{\text{chicken ing}} + ICR_{\text{egg ing}}$$

ICR_{on}	=	Cumulative Incremental Cancer Risk for the On-site Residential Receptor (unitless)
$ICR_{\text{soil ing}}$	=	Incremental Cancer Risk for the Soil Ingestion Pathway (unitless)
$ICR_{\text{dust inh}}$	=	Incremental Cancer Risk for the Dust Inhalation Pathway (unitless)
ICR_{dermal}	=	Incremental Cancer Risk for the Dermal Contact Pathway (unitless)
$ICR_{\text{fruit \& veg ing}}$	=	Incremental Cancer Risk for the Garden Fruit and Vegetable Ingestion Pathway (unitless)
$ICR_{\text{chicken ing}}$	=	Incremental Cancer Risk for the Chicken Ingestion Pathway (unitless)
$ICR_{\text{egg ing}}$	=	Incremental Cancer Risk for the Egg Ingestion Pathway (unitless)

$$ICR_{off} = ICR_{\text{soil ing}} + ICR_{\text{dust inh}} + ICR_{\text{dermal}} + ICR_{\text{fruit \& veg ing}}$$

ICR_{off}	=	Cumulative Incremental Cancer Risk for the Off-site Residential Receptor (unitless)
$ICR_{\text{soil ing}}$	=	Incremental Cancer Risk for the Soil Ingestion Pathway (unitless)
$ICR_{\text{dust inh}}$	=	Incremental Cancer Risk for the Dust Inhalation Pathway (unitless)
ICR_{dermal}	=	Incremental Cancer Risk for the Dermal Contact Pathway (unitless)
$ICR_{\text{fruit \& veg ing}}$	=	Incremental Cancer Risk for the Garden Fruit and Vegetable Ingestion Pathway (unitless)

Incremental Cancer Risk - Incidental Soil Ingestion

$$ICR_{soiling} = \frac{\left(C_{s,i} \times \left[\left(\frac{IR_{s,site,a} \times EF_a \times ED_{a,carc}}{BW_a}\right) + \left(\frac{IR_{s,site,c} \times EF_c \times ED_c}{BW_c}\right)\right] \times SF_{ing,i} \times 10^{-6}\, kg/mg\right)}{AT_{carc}}$$

$ICR_{soil\ ing}$	=	Incremental Cancer Risk for the Soil Ingestion Pathway (unitless)
$C_{s,i}$	=	Concentration of Contaminant *i* in Exposure Area Soil (mg/kg soil)
$IR_{s,site,a}$	=	Site-Specific Soil Ingestion Rate, Adult (mg/d)
$IR_{s,site,c}$	=	Site-Specific Soil Ingestion Rate, Child (mg/d)
EF_a	=	Exposure Frequency, Adult (d/yr)
EF_c	=	Exposure Frequency, Child (d/yr)
$ED_{a,carc}$	=	Exposure Duration, Adult (cancer) [yr]
ED_c	=	Exposure Duration, Child (yr)
BW_a	=	Body Weight, Adult (kg)
BW_c	=	Body Weight, Child (kg)
$SF_{ing,i}$	=	Ingestion Slope Factor, contaminant *i* $(mg/kg\text{-}d)^{-1}$
AT_{carc}	=	Averaging Time (cancer) [d]

$$IR_{s,site} = IR_s \times \left\{\frac{\left(E_{out} \times T_{site,out}\right) + \left(\frac{T_{site,in}}{E_{out}}\right)}{\left(E_{out} \times T_{out}\right) + \left[\frac{\left(T_{in} - T_{sleep}\right)}{E_{out}}\right]}\right\}$$

$IR_{s,site}$	=	Site-Specific Soil Ingestion Rate (mg/d)
IR_s	=	Incidental Soil Ingestion Rate (mg/d)
E_{out}	=	Outdoor Activity Soil Loading Enrichment Factor (unitless)
$T_{site,out}$	=	Time Onsite Spent Outdoors (hr/d)
$T_{site,in}$	=	Time Onsite Spent Indoors (hr/d)
T_{out}	=	Total Time Spent Outdoors (hr/d)
T_{in}	=	Total Time Spent Indoors (hr/d)
T_{sleep}	=	Time Spent Sleeping or Napping (hr/d)

Incremental Cancer Risk - Dust Inhalation

$$ICR_{\text{dust inh}} = \frac{\left(C_{s,i} \times \left[\left(\frac{InhR_a \times ET_a \times EF_a \times ED_{a,carc}}{BW_a}\right) + \left(\frac{InhR_c \times ET_c \times EF_c \times ED_c}{BW_c}\right)\right] \times SF_{inh,i}\right)}{\left(PEF \times AT_{carc}\right)}$$

$ICR_{\text{dust inh}}$	=	Incremental Cancer Risk for the Dust Inhalation Pathway (unitless)
$C_{s,i}$	=	Concentration of Contaminant *i* in Exposure Area Soil (mg/kg soil)
$InhR_a$	=	Inhalation Rate, Adult (m^3/hr)
$InhR_c$	=	Inhalation Rate, Child (m^3/hr)
ET_a	=	Exposure Time, Adult (d/yr)
ET_c	=	Exposure Time, Child (d/yr)
EF_a	=	Exposure Frequency, Adult (d/yr)
EF_c	=	Exposure Frequency, Child (d/yr)
$ED_{a,\text{ carc}}$	=	Exposure Duration, Adult (cancer) [yr]
ED_c	=	Exposure Duration, Child (yr)
BW_a	=	Body Weight, Adult (kg)
BW_c	=	Body Weight, Child (kg)
$SF_{inh,I}$	=	Inhalation Slope Factor, contaminant *i* $(mg/kg\text{-}d)^{-1}$
PEF	=	Particulate Emission Factor (m^3/kg)
AT_{carc}	=	Averaging Time (cancer) [d]

$$PEF = \frac{\left(Q/C \times 3600\,\text{sec}/hr\right)}{\left(0.036 \times (1-v) \times \left(\frac{U_m}{U_{t-7}}\right)^3 \times F(x)\right)}$$

PEF	=	Particulate Emission Factor (m^3/kg)
Q/C	=	Inverse of Mean Concentration at the Center of a Square Source Area (g/m^2-sec per kg/m^3)
v	=	Fraction of Vegetative Cover (unitless)
U_m	=	Mean Annual Windspeed (m/sec)
U_{t-7}	=	Equivalent Threshold Wind Speed at 7 m (m/s)
F(x)	=	Function dependent on U_m/U_{t-7}

$$U_{t-7} = \left(\frac{U_t}{0.4}\right) \times \ln\left(\frac{z}{z_0}\right)$$

U_{t-7}	=	Equivalent Threshold Wind Speed at 7 m (m/s)
U_t	=	Threshold Friction Velocity (m/s)
z	=	Height Above Surface (m)
z_0	=	Surface Roughness Height (m)

Incremental Cancer Risk - Dermal Contact

$$ICR_{dermal} = \frac{\left(C_{s,i} \times ABS_i \times \left[\left(\frac{AF_{adj,a} \times EF_{derm,a} \times ED_{a,carc}}{BW_a}\right) + \left(\frac{AF_{adj,c} \times EF_{derm,c} \times ED_c}{BW_c}\right)\right] \times \left(\frac{SF_{ing,i}}{F_{oral,i}}\right) \times 10^{-6}\, kg/mg\right)}{AT_{carc}}$$

ICR_{dermal}	=	Incremental Cancer Risk for the Dermal Contact Pathway (unitless)
$C_{s,i}$	=	Concentration of Contaminant *i* in Exposure Area Soil (mg/kg soil)
ABS_i	=	Dermal Absorption Factor, contaminant *i* (unitless)
$AF_{adj,\,a}$	=	Surface-Area-Adjusted Adherence Factor, Adult (mg/event)
$AF_{adj,\,c}$	=	Surface-Area-Adjusted Adherence Factor, Child (mg/event)
$EF_{derm,\,a}$	=	Exposure Frequency for Dermal Contact with Soil, Adult (event/yr)
$EF_{derm,\,c}$	=	Exposure Frequency for Dermal Contact with Soil, Child (event/yr)
$ED_{a,\,carc}$	=	Exposure Duration, Adult (cancer) [yr]
ED_c	=	Exposure Duration, Child (yr)
BW_a	=	Body Weight, Adult (kg)
BW_c	=	Body Weight, Child (kg)
$SF_{ing,i}$	=	Ingestion Slope Factor, contaminant *i* $(mg/kg\text{-}d)^{-1}$
$F_{oral,i}$	=	Oral Absorption Factor for Contaminant *i* (unitless)
AT_{carc}	=	Averaging Time (cancer) [d]

$$AF_{adj} = (SA_h \times AF_h) + (SA_a \times AF_a) + (SA_l \times AF_l)$$

AF_{adj}	=	Surface-Area-Adjusted Adherence Factor (mg/event)
SA_h	=	Surface Area of Hand (cm^2)
SA_a	=	Surface Area of Arm (cm^2)
SA_l	=	Surface Area of Leg (cm^2)
AF_h	=	Soil Adherence Factor of Hand (mg/cm^2-event)
AF_a	=	Soil Adherence Factor of Arm (mg/cm^2-event)
AF_l	=	Soil Adherence Factor of Leg (mg/cm^2-event)

Incremental Cancer Risk - Fruit and Vegetable Ingestion

$$ICR_{fruit\,\&\,veg\,ing} = \frac{\left(C_{s,i} \times K_{p-s,i} \times \left[\left(IR_v \times \frac{depth_{cz}}{depth\_root_v}\right) + \left(IR_f \times \frac{depth_{cz}}{depth\_root_f}\right)\right] \times EF_{food} \times \left(ED_{a,carc} + ED_c\right) \times SF_{ing,i} \times 10^{-3}\,kg/g\right)}{AT_{carc}}$$

$ICR_{fruit\,\&\,veg\,ing}$	=	Incremental Cancer Risk for the Garden Fruit and Vegetable Ingestion Pathway (unitless)
$C_{s,i}$	=	Concentration of Contaminant *i* in Exposure Area Soil (mg/kg soil)
$K_{p-s,i}$	=	Plant:Soil Concentration Ratio, contaminant *i* (mg/kg wet weight plant per mg/kg soil)*
IR_v	=	Ingestion Rate of Home-Grown Vegetables (g/kg-d)
IR_f	=	Ingestion Rate of Home-Grown Fruits (g/kg-d)
$depth_{cz}$	=	Depth of Contaminated Zone (cm)
$depth\_root_v$	=	Effective Rooting Depth of Garden Vegetables (cm)
$depth\_root_f$	=	Effective Rooting Depth of Fruit Trees (cm)
EF_{food}	=	Exposure Frequency for Ingesting Food Products (d/yr)
$ED_{a,carc}$	=	Exposure Duration, Adult (cancer) [yr]
ED_c	=	Exposure Duration, Child (yr)
$SF_{ing,i}$	=	Ingestion Slope Factor, contaminant *i* $(mg/kg\text{-}d)^{-1}$
AT_{carc}	=	Averaging Time (cancer) [d]

$$K_{p-s,i} = B_r \times CvF_p$$

$K_{p-s,i}$	=	plant:soil concentration ratio, contaminant I (mg/kg wet weight plant per mg/kg soil)*
B_r	=	Baes Soil-to-Root Transfer Factor (mg/kg dry weight plant per mg/kg soil)
CvF_p	=	Conversion Factor for Fruits (unitless)

* The fruit and vegetable ingestion equation presented in the 2001 HHRA memo has an error. Kp-s is represented as mg/kg dry weight plant per mg/kg soil, and is multiplied by the conversion factor used to convert from dry to wet weight. However, in spreadsheets provided to Chino on February 9, 2001, (R. Perona, pers. comm) Kp-s is defined as the Baes transfer factor (mg/kg dry weight) multiplied by the conversion factor used to convert from dry to wet weight. Therefore, the equation appears to include the conversion factor twice. This equation presents the corrected algorithm.

Incremental Cancer Risk - Egg Ingestion

$$ICR_{egging} = \frac{\left(C_{s,i} \times TF_{s-e,i} \times UR_f \times F_{s-f} \times IR_e \times EF_{food} \times \left(ED_{a,carc} + ED_c\right) \times SF_{ing,i} \times 10^{-3}\, kg/g\right)}{AT_{carc}}$$

$ICR_{egg\ ing}$	=	Hazard Quotient for the Egg Ingestion Pathway (unitless)
$C_{s,i}$	=	Concentration of Contaminant *i* in Exposure Area Soil (mg/kg soil)
$TF_{s-e,i}$	=	Soil-Egg Transfer Factor, contaminant I (mg/kg egg per mg/d)
UR_f	=	Uptake Rate of Dry Feed (kg/d)
$F_{s,f}$	=	Fraction of Soil in Chicken Feed (unitless)
IR_e	=	Egg Ingestion Rate (g/kg-d)
$ED_{a,\ carc}$	=	Exposure Duration, Adult (cancer) [yr]
ED_c	=	Exposure Duration, Child (yr)
ED_{nc}	=	Exposure Duration (noncancer) [yr]
$SF_{ing,i}$	=	Ingestion Slope Factor, contaminant *i* $(mg/kg\text{-}d)^{-1}$
AT_{carc}	=	Averaging Time (cancer) [d]

* The egg ingestion equation presented in the 2001 HHRA memo has an error. Neptune (R. Perona, pers. comm) confirmed that Kf-s was renamed Fs,f, and Kf-s (and its definition) should be deleted from the equation's list of parameters. The corrected version of the equation is shown here.

** The units shown in the 2001 HHRA memo for the egg ingestion rate (kg/yr) appear to be in error. A dimensional analysis shows that the units for this parameter presented in the RME references table of the memo (g/kg-d) are mathematically correct. These units have been assumed, and are presented here.